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REVIEW PAPER

Auxin and above-ground meristems

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Abstract

In contrast to animals, plants maintain life-long post-embryonic organogenesis from specialized tissues termed meristems. Shoot meristems give rise to all aerial tissues and are precisely regulated to balance stem cell renewal and differentiation. The phytohormone auxin has a dynamic and differential distribution within shoot meristems and during shoot meristem formation. Polar auxin transport and local auxin biosynthesis lead to auxin maxima and minima to direct cell fate specification, which are critical for meristem formation, lateral organ formation, and lateral organ patterning. In recent years, feedback regulatory loops of auxin transport and signaling have emerged as major determinants of the self-organizing properties of shoot meristems. Systems biology approaches, which involve molecular genetics, live imaging, and computational modeling, have become increasingly important to unravel the function of auxin signaling in shoot meristems.

Keywords: Adventitious meristem, auxin, axillary meristem, floral meristem, patterning, shoot apical meristem.

Introduction

Distinct from animals, plants maintain continuous organogenesis from stem cells located in meristems throughout their life cycle (Barton, 2010; Stahl and Simon, 2010; Aichinger *et al.*, 2012; Murray *et al.*, 2012; Perales and Reddy, 2012). The above-ground aerial organs of the plants come from the shoot apical meristem (SAM) and the underground organs come from the root apical meristem. Both meristems contain a mass of stem cells in the center, which divide to maintain themselves and to provide cells that make up new organ primordia.

The establishment of the SAM takes place during embryogenesis. Post-embryonically, axillary meristems (AMs) form in the leaf axil to enable branching. Although formed post-embryonically, AMs share a similar structure and function with the embryonically formed SAM. After transition into reproductive growth, the SAM is transformed into the inflorescence meristem (IM), and contributes to plant reproductive success. Floral meristems (FMs) are generated from the IM and directly produce a limited number of floral organs, including sepals, petals, stamens, and carpels. Distinct from the IM, FMs only exhibit transient stem cell activity. Molecular marker expression suggests that the FM is a specialized AM whereas the leaf is specialized into a bract or cryptic bract (Long and Barton, 2000). In fact, it has long been proposed, after von Goethe (1790), that a flower can be considered as a compressed and determinate shoot. Although with distinctions, these different types of shoot meristems share similar structures and molecular signatures.

Auxin is a major plant hormone that is involved in various developmental processes. In addition to biosynthesis and degradation, polar auxin transport, the directional cell to cell transport of auxin, is a major process determining the spatial auxin distribution. Among the several transmembrane efflux and influx carriers, the PIN-FORMED (PIN) family auxin efflux carriers are particularly important for the generation of morphogenetic auxin gradients (Okada *et al.*, 1991; Leyser, 2010; Adamowski and Friml, 2015). A number of excellent recent reviews have focused on the biosynthesis, transport, perception, and signaling of auxin (Zhao, 2012; Guilfoyle, 2015; Lavy and Estelle, 2016), including those published in this special issue. In this review, we limit ourselves to the roles of auxin on shoot meristems.

Organization and genetic regulation of shoot meristems

The SAM is initially formed during embryogenesis, when the basic body architecture of a plant is established. In dicotyledonous plants, such as *Arabidopsis,* the SAM is established in the apex between two cotyledons. During post-embryonic development, the SAM generates stems, leaves, and floral organs in a set pattern while it maintains a pool of undifferentiated cells in the center (Steeves and Sussex, 1989). The structure of the SAM is generally well conserved, and can be divided into an external tunica layer and an inner corpus region. These two regions are very different at the cellular level: whereas cells of the corpus divide without a preferential cell division plane, cells of the tunica mostly divide perpendicular to the surface (or anticlinally). The anticlinical division pattern of tunica cells generates a layered structure with daughter cells remaining in the same layer as their parents.

The SAM can also be divided into three domains based on function, the central zone (CZ), the peripheral zone (PZ), and the rib meristem (RM) (Fig. 1) (Steeves and Sussex, 1989). The CZ is located at the apex of the SAM, and harbors pluripotent stem cells. Stem cells in the CZ divide slowly to replenish themselves. Some of the stem cell progenies are displaced from the CZ into the surrounding PZ, where cells divide rapidly to form organ primordia. The RZ is located underneath the CZ and contains cells that are determined to form the internal tissue of the stem. Between the CZ and the RZ are

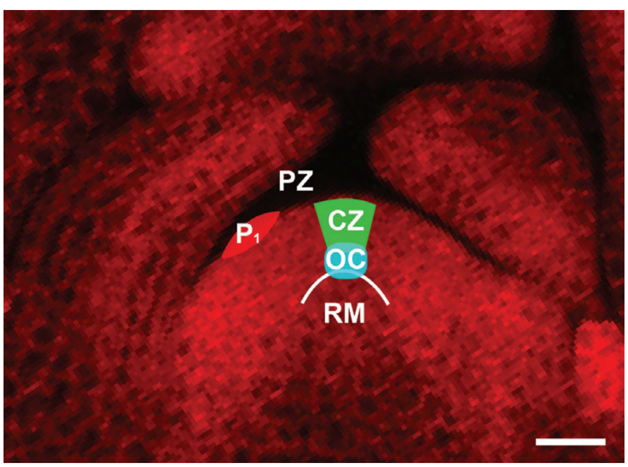

Fig. 1. The SAM of *Arabidopsis thaliana*. Longitudinal section of a vegetative SAM showing functional zones. At the SAM apex, the central zone (CZ) contains stem cells, the organizing center (OC) induces stem cells in the CZ, and primordia are initiated in the peripheral zone (PZ). The rib meristem (RM) produces the stem. Scale bar=20 µm.

a small group of quiescent cells making up the organizing center (OC), which maintains the stem cell niche above it. The zonation into functional domains in the SAM is dynamic (Laufs *et al.*, 1998), as shown by molecular markers (Reddy and Meyerowitz, 2005; Müller *et al.*, 2006).

The homeodomain transcription factor WUSCHEL (WUS) is expressed in the OC to maintain stem cells in the CZ (Mayer *et al.*, 1998). WUS migrates to the CZ to activate the expression of the negative regulator *CLAVATA3* (*CLV3*), which encodes a secreted peptide (Fletcher *et al.*, 1999; Yadav *et al.*, 2011; Daum *et al.*, 2014). The secreted extracellular CLV3 peptide activates CLV1, a transmembrane repeat receptor kinase expressed in the OC, to inhibit *WUS* expression (Clark *et al.*, 1997; Ogawa *et al.*, 2008). Thus, the WUS–CLV feedback loop forms a self-correcting mechanism that maintains a stem cell pool of constant size (Brand *et al.*, 2000; Schoof *et al.*, 2000; Somssich *et al.*, 2016). Together with *WUS*, the class 1 *KNOTTED*-like homeobox (*KNOX1*) gene is critical for maintenance of the SAM, and the expression of *KNOX1* is inhibited by *ASYMMETRIC LEAVES1*/*ROUGH SHEATH2*/*PHANTASTICA* (*ARP*) genes expressed in leaves (Hay and Tsiantis, 2010).

Auxin and the shoot apical meristem

A prominent function of auxin in the SAM is to promote primordium formation, either leaf primordia during the vegetative stage or floral primordia during the reproductive stage. Auxin microapplication experiments have suggested that a local auxin maximum is necessary and sufficient to trigger primordium initiation in the PZ of the SAM (Reinhardt *et al.*, 2000). An auxin maximum causes local interference with cell wall anisotropy and a limited reduction in wall stiffness, which promote organogenesis (Sassi *et al.*, 2014). Furthermore, polar auxin transport leads to auxin accumulation to establish local auxin maxima, which specify incipient primordia. PIN1 is expressed in the epidermal layer and the provascular cells in the SAM. The formation of local auxin maxima results from coordinated PIN1 polarity of each cell, which is highly dynamic and stereotypic (Reinhardt *et al.*, 2003; Heisler *et al.*, 2005).

By assuming that cells localize PIN1 towards the neighboring cell with a higher intracellular auxin concentration (up the gradient) (Jönsson *et al.*, 2006; Smith *et al.*, 2006), or that PIN1 localization to cell membranes is in proportion to the auxin flux rate across the membrane (flux based) (Mitchison, 1980; Stoma *et al.*, 2008), computational models have been able to explain the self-organized arrangement of primordia at the SAM, namely phyllotaxis. We refer the readers to recent reviews on phyllotaxis for a more exhaustive view (Sassi and Vernoux, 2013; Traas, 2013; Galvan-Ampudia *et al.*, 2016). In addition, recent ground-breaking studies have identified additional regulatory mechanisms of phyllotaxis. It is established that MONOPTEROS (MP; also called ARF5) is a key transcription factor mediating the auxin transcriptional response (Lavy and Estelle, 2016). A recent study has provided the first solid experimental support for the above-mentioned phyllotaxis models. *MP* expression is controlled in an auxin-dependent self-activating way. Furthermore, localized MP activity orients PIN1 polarity non-cell autonomously to promote local auxin maxima formation and organ formation (Bhatia *et al.*, 2016). In addition, auxin induces *PIN1* expression through MP (Krogan *et al.*, 2016). In combination with the PIN1-dependent formation of auxin maxima, these positive feedback loops are probably critical for the self-organization properties of the SAM (Fig. 2). Auxin directly activates expression of the cytokinin signaling inhibitor ARABIDOPSIS HISTIDINE PHOSPHOTRANSFER PROTEIN 6 (AHP6). Further intercellular movement of AHP6 generates inhibitory fields of cytokinin signaling and contributes to the robustness of phyllotaxis (Besnard *et al.*, 2014).

Since the 1950s, microsurgical experiments have suggested that the SAM also promotes leaf adaxial–abaxial (dorsoventral) patterning (Sussex, 1951; Reinhardt *et al.*, 2005; Kuhlemeier and Timmermans, 2016). A recent study suggested that polar auxin transport can explain the meristem-derived leaf polarity signal (Qi *et al.*, 2014). A leaf primordium initiates following formation of an auxin maximum. After bulging outward, subsequent auxin transport from the newly formed primordia back to the SAM leads to differential auxin concentrations within leaf primordia, which promote leaf polarity patterning. Thus, it is not a positive signal from the SAM, but departure of auxin from primordia to the SAM, that delivers polarity information—opposite to the original proposal.

On the other hand, it is less clear whether auxin regulates the formation or homeostasis of the SAM. Whereas a number of auxin biosynthesis, transport, and signaling mutants have defects in lateral organ formation (Okada *et al.*, 1991; Przemeck *et al.*, 1996; Vernoux *et al.*, 2000), or have compromised phyllotactic patterning (Cheng *et al.*, 2007; Guenot *et al.*, 2012; Pinon *et al.*, 2013), the SAM structure is not severely affected (Vernoux *et al.*, 2000). Auxin signaling sensors DR5 and DII indicate that the SAM is generally low in

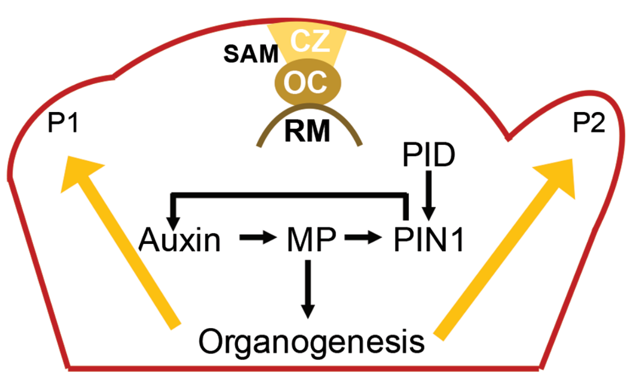

Fig. 2. Conceptual summary of auxin regulation in SAM function. SAM is divided into three functional zones, namely the central zone (CZ), the rib meristerm (RM), and the peripheral zone (PZ), where primordia initiate. Auxin regulates organogenesis, such as leaf primordium development and flower development, through one of its downstream response factors, MP. Auxin distribution is determined by PIN1-mediated auxin efflux, while PIN1 orientation is regulated by its phosphorylation by PID. In turn, MP also affects the polarity of PIN1 localization in a non-cell-autonomous way.

auxin signaling (Benkova *et al.*, 2003; Brunoud *et al.*, 2012; Vernoux *et al.*, 2011), except for periodic formation of auxin maxima in the epidermal layer. Given that high auxin levels inhibit AM activity and adventitious shoot formation (Domagalska and Leyser, 2011; Su *et al.*, 2011), auxin may also have a negative effect on embryonic SAM formation and/or homeostasis. Detailed analyses of auxin transport and signaling mutants have supported this proposal (Schuetz *et al.*, 2008). Also, it has been reported that MP inhibits the expression of two A-type ARABIDOPSIS RESPONSE REGULATOR (ARR) genes, ARR7 and ARR15 (Zhao *et al.*, 2010). As ARR7 and ARR15 negatively regulate SAM size, auxin signaling could promote SAM activity. It remains to be resolved whether this A-type ARR-mediated regulation is a main effect, or a compensatory feedback.

Axillary meristem initiation requires an auxin minimum

A typical seed plant can have multiple growth axes, each with an AM. Except for the main growth axis, all secondary growth axes are established by AMs. AMs are derived from the SAM: the SAM continuously produces phytomers along the stem, each of which contains a leaf, an AM, and an internode. AMs reside in or near leaf axils and function as new SAMs to make a ramifying shoot. The SAM and AMs together contribute to the overall growth and architecture of the plant (Wang and Li, 2008).

AM formation, or initiation, involves the establishment of the stem cell niche in the boundary region between the stem and the leaf. AMs share similar structures and gene expression with the vegetative SAM (Schmitz and Theres, 2005). Nevertheless, genetic studies have identified several genes that specifically regulate AM initiation, indicating that AM initiation is different from that of the embryonic SAM (Wang *et al.*, 2016; Yang and Jiao, 2016). Also, there are differences in AM and SAM regarding gene expression (Serrano-Mislata *et al.*, 2016).

Recent studies have shown that a low auxin environment is critical for AM initiation (Fig. 3) (Q. Wang *et al.*, 2014; Y. Wang *et al.*, 2014). As mentioned above, leaf formation is accompanied by complex and dynamic changes in PIN1 orientation in the SAM. PIN1-mediated auxin transport toward a convergence point acts to trigger leaf formation. However, PIN1 polarity reverses orientation back towards the meristem center (Heisler *et al.*, 2005; Bayer *et al.*, 2009; Qi *et al.*, 2014; Q. Wang *et al.*, 2014; Y. Wang *et al.*, 2014). Thus, auxin is depleted from the boundary region and an auxin minimum is created. PINOID (PID) regulates the intracellular localization of the PIN auxin efflux carriers (Friml *et al.*, 2004), and is enriched in the leaf axil (Landrein *et al.*, 2015). Therefore, an auxin minimum in the leaf axil relies on PIN1 and PID. The local auxin minimum is necessary for AM initiation, as *pin1* and *pid* mutants are defective in AM formation. Consistently, ectopic production of auxin by an auxin biosynthetic gene *iaaM* in the boundary region results in aberrant AM formation (Q. Wang *et al.*, 2014; Y. Wang *et al.*, 2014). In contrast, expressing an undegradable version of the AUX/IAA repressor BODENLOS/IAA12 in the leaf axils largely restores AM initiation in the *pid-9* mutant and can further cause ectopic formation of AMs in the cotyledon axils (Q. Wang *et al.*, 2014).

The local auxin minimum is critical for the maintenance of a meristematic cell population in the leaf axil (Fig. 3). A recent study has shown that a group of cells sustain expression of the meristem marker *STM*, and AMs are formed only from the progeny of these *STM*-expressing cells (Shi *et al.*, 2016). It has been demonstrated that the continuous expression of *STM*, but not other tested AM initiation genes, requires the auxin minimum. Later in development, a transient cytokinin signal pulse appears in the leaf axil, and is required for AM initiation (Y. Wang *et al.*, 2014; Wang *et al.*, 2017). The cytokinin signaling also requires the prior auxin minimum (Y. Wang *et al.*, 2014).

However, after floral transition, a different auxin regulation mechanism might be employed. A recent study using live cell imaging reached the conclusion that cells expressing the auxin reporter DR5 form the axillary buds (Burian *et al.*, 2016). Notably, this study tracked cauline leaf axillary bud formation. Cauline leaves form after floral transition, and cauline leaf buds have a mixed fate of an AM and FM (Hempel and Feldman, 1995). Because auxin promotes FM formation (see below), it is possible that the auxin minimum is no longer required for cauline leaf axillary bud formation.

Auxin and adventitious meristem formation

Adventitious meristems form buds at places other than the SAM at the shoot apex or AMs at the leaf axil. They may develop on roots or leaves, or on shoots as a new growth axis. Plants have a profound capacity for regeneration, which also relies on adventitious meristem formation. Adventitious shoots can form naturally, especially from cut surfaces. Early findings based on work with plant tissue culture indicate that a low auxin/cytokinin ratio induces adventitious shoot regeneration, a high ratio induces root induction, and an intermediate ratio induces disorganized cell masses, called calli (Skoog and Miller, 1957). Thus, phytohormonal regulation is actively involved in adventitious meristem formation. Recent breakthrough experiments have shown that rather than dedifferentiation, calli form predominantly from an existing stem cell population (Atta *et al.*, 2009; Sugimoto *et al.*, 2010). These specific cells surround the vasculature, and are termed xylem pole pericycle cells in roots and hypocotyls, and pericycle-like cells in other organs with shared maker gene expression (Fig. 4A). The pericycle and pericycle-like cells are also responsible for lateral root and adventitious root formation (Hu and Xu, 2016). Gene expression indicates that callus resembles root meristem, rather than SAM or embryonic tissues. When provided with a low auxin/cytokinin ratio, callus or even existing lateral and adventitious root meristems transdifferentiate into adventitious shoot meristems (Fig. 4B).

The above framework for adventitious meristem formation provides an explanation for the importance of the auxin/cytokinin ratio (Fig. 4). It has been well established that lateral root and adventitious root formation require auxin maxima (Chen *et al.*, 2016; Möller *et al.*, 2017), consistent with the requirement for a high auxin/cytokinin ratio for root formation. Following callus formation, a local auxin gradient is established, and *WUS* expression is established in cells with low auxin signaling (Gordon *et al.*, 2007; Cheng *et al.*, 2013), similar to AM initiation (Q. Wang *et al.*, 2014; Y. Wang *et al.*, 2014). The spatial auxin distribution relies on both biosynthesis and polar auxin transport (Cheng *et al.*, 2013). The activation of cytokinin signaling subsequently promotes *WUS* expression to establish a new stem shoot cell niche (Meng *et al.*, 2017; Zhang *et al.*, 2017), which is again highly similar to AM initiation (Wang *et al.*, 2017). Taken together, adventitious shoot meristem formation shares similarity with AM

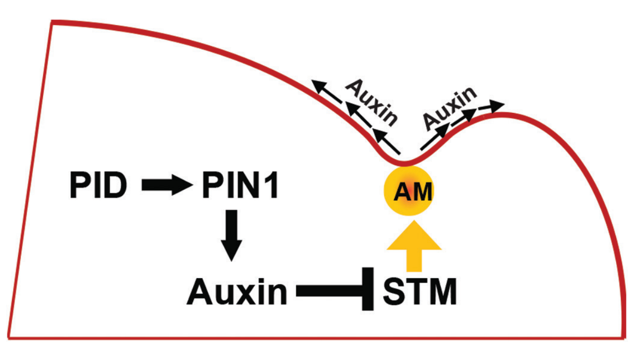

Fig. 3. Conceptual summary of auxin regulation of AM formation. During leaf development, an auxin minimum (orange) is first observed at the leaf axil due to PIN1-mediated auxin flow, which is required for proper AM initiation during vegetative development.

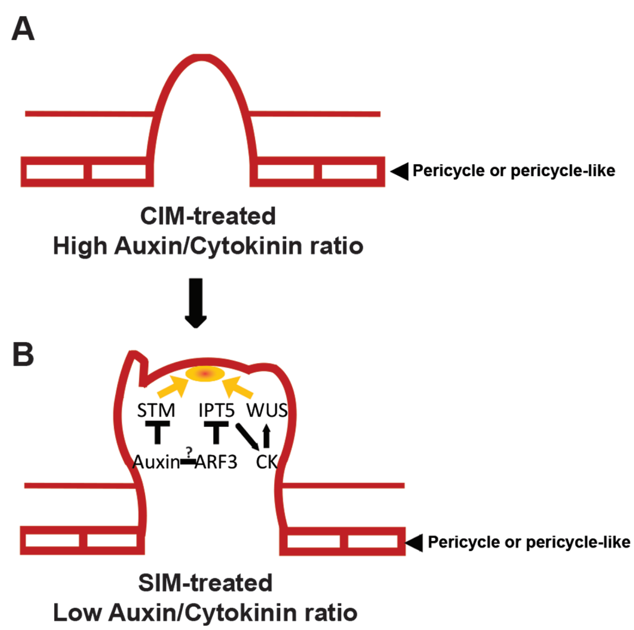

Fig. 4. Schematic diagram of auxin regulation of adventitious meristem formation on shoot-inducing medium (SIM). (A) Calli formed on high-auxin callus-inducing medium (CIM) first develop as lateral root initials. (B) When induced on low-auxin SIM, adventitious meristems are formed. Auxin is known to inhibit *STM* expression, and therefore inhibit adventitious meristem formation. In contrast, cytokinin promotes *WUS* expression, which together with *STM* further specifies the stem cell niche in adventitious shoots.

initiation, including the *de novo WUS* activation mechanism. In addition, adventitious meristem and AM derive from different pre-existing stem cell populations (Sugimoto *et al.*, 2010; Shi *et al.*, 2016).

A recent study has reported the formation of another type of adventitious meristem formed on tomato leaves (Rossmann *et al.*, 2015). Adventitious meristems can initiate at the base of tomato leaflets, a property which is shared with some other plant species. Such ectopic buds can initiate new growth axes and even vegetative propagation, as in *Bryophyllum daigremontianum*, commonly called mother-of-thousands. Genetic analysis indicated that adventitious meristem formation at the base of tomato leaflets is regulated by the same set of genes used in AM initiation (Rossmann *et al.*, 2015). Given that auxin minima precede leaflet base formation (Berger *et al.*, 2009; Koenig *et al.*, 2009), it is conceivable that an auxin minimum is required for such adventitious meristem formation as in AM initiation. These adventitious shoot meristems from leaflets may also derive from *STM*-expressing meristematic cells at the base of tomato leaflets (Kim *et al.*, 2003).

Auxin promotes reproductive meristems

After transition to flowering, the SAM becomes an IM and forms flower primordia in the periphery. Similar to the vegetative SAM, initiation of a flower primordium requires a local auxin maximum at the PZ. Flower primordia form transient FMs that contain stem cells to form floral organs. Although the FM has been considered as a specialized AM (Long and Barton, 2000), auxin has very different effects on FM from those on AM (Fig. 3).

Auxin is required for FM formation (Fig. 5). As in the vegetative SAM, polar auxin transport leads to dynamic auxin distribution in the IM. Mutations in *PIN1* result in naked inflorescence stalks lacking flowers (Okada *et al.*, 1991). Similarly, mutations in *PID*, which regulates PIN localization, also lead to naked pins without FMs (Bennett *et al.*, 1995).

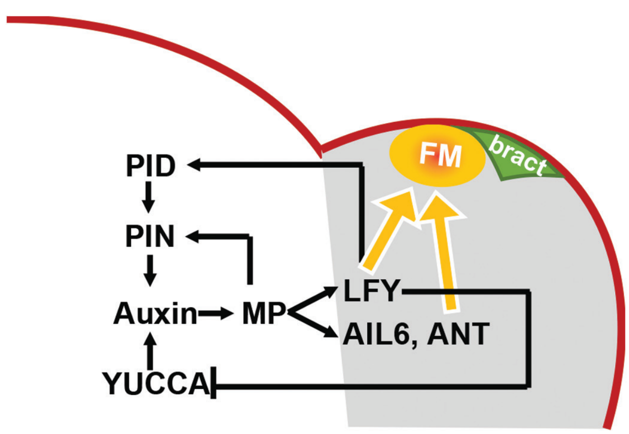

Fig. 5. Conceptual summary of auxin regulation of FM formation. During reproductive development, auxin (red) promotes FM formation at least partially through activating *LFY*, *AIL6*, *ANT*, and other transcriptional regulators. A major auxin response factor, MP, directly binds to and activates *LFY*, *AIL6*, and *ANT*. In turn, LFY also regulates auxin production and distribution through impacting *YUCCA* and *PID* expression.

Besides the genes that play roles in creating auxin maxima during primordium initiation, genes that have central roles in auxin response are also critical for FM formation. Among them, *MP* is of primary importance in FM formation. Like *pin1* and *pid* mutants, *mp* mutants form naked stalks without flowers (Przemeck *et al.*, 1996). In contrast, leaves still form in the above single mutants, and a naked vegetative SAM forms only when both polar auxin transport (as in *pin1* or *pid*) and auxin signaling (as in *mp*) are disrupted (Schuetz *et al.*, 2008). This difference implies that FMs could be more sensitive to auxin than leaf primordia. Alternatively, PIN1 and MP may have more dominant effects on polar auxin transport and signaling, respectively, in the IM. It is also conceivable that there is more auxin in the vegetative meristem.

Upon floral transition, *LEAFY* (*LFY*), a master regulator of reproductive growth, is activated by auxin (Yamaguchi *et al.*, 2013). LFY encodes a transcription factor that specifies floral fate of meristems and is a master co-ordinator of the entire floral network (Moyroud *et al.*, 2010; Siriwardana and Lamb, 2012). In *Arabidopsis,* complete loss in *LFY* functions causes partial transformation of flowers into inflorescence shoots, while in *Antirrhinum* (snap dragon), it results in more complete transformation of flowers into inflorescence shoots (Coen *et al.*, 1990; Weigel and Meyerowitz, 1993; Prusinkiewicz *et al.*, 2007). *LFY* is expressed in FMs and repressed in the apical IM. It was found that *LFY* expression is defined by auxin maxima (Vernoux *et al.*, 2000; Li *et al.*, 2013). In *pin1* mutants, *LFY* expression was strongly affected in the PZ, probably due to altered cell identity in the region. Furthermore, a recent study showed that MP directly activates *LFY* expression (Yamaguchi *et al.*, 2013). The *LFY* promoter region contains several evolutionarily conserved auxin response element (AuxRE) core motifs, to which MP directly binds. In a hypomorphic *mp* mutant, *LFY* mRNA and protein levels were greatly reduced, especially in the PZ of the IM, reminiscent of what happens in *pin1* mutants. Treatment with auxin or its analog results in a rapid and robust increase in *LFY* expression, whereas treatment with the auxin transport inhibitor 1-*N*-naphthylphthalamic acid (NPA) results in a decrease in *LFY* expression.

In addition to *LFY*, two other transcription factor-encoding genes, *AINTEGUMENTA* (*ANT*) and *ANT-LIKE6/PLETHORA3* (*AIL6/PLT3*), play important roles in mediating auxin responses during FM specification. Both *ANT* and *AIL6* are also direct targets of MP (Yamaguchi *et al.*, 2013), and they further activate *LFY* expression (Yamaguchi *et al.*, 2016). Taken together, auxin activates *LFY* and other key regulators to direct cell fate reprogramming from transient amplifying to primordium founder cell fate during FM formation. A chromatin state switch is involved in the auxin activation of FM fate (Wu *et al.*, 2015). MP as a transcription factor recruits SWI/SNF chromatin-remodeling ATPases to increase DNA accessibility for target gene induction and further enable FM formation.

Whereas auxin regulates *LFY* expression, *LFY* feeds back to the auxin pathway (Li *et al.*, 2013; Yamaguchi *et al.*, 2013). In particular, LFY directly modulates auxin transport by promoting *PID* expression (Yamaguchi *et al.*, 2013). On the other hand, LFY inhibits auxin biosynthesis by suppressing *YUC* expression (Li *et al.*, 2013). Therefore, FM formation is under the control of both auxin and transcriptional master regulators.

Although extensively studied, the auxin regulation of FM formation still warrants further scrutiny. Detailed analysis of geometry changes and gene expression dynamics suggests that FM forms on the axil of a putative rudimentary bract in *Arabidopsis* (Kwiatkowska, 2006; Chandler and Werr, 2014). The initial auxin convergence in the IM is probably associated with the rudimentary bract formation, while the associated FM forms later. Hence low auxin levels may also be associated with FM formation, as they are for AMs. In fact, *LFY* is expressed not only in FMs but also in leaves during the transition to flowering (Liljegren *et al.*, 1999). *ANT*, *FIL*, and *AIL6* are also associated with leaf/bract formation and therefore could be only indirectly involved in the flower meristem. Thus, auxin might more directly influence formation of the subtending rudimentary bract, rather than the FM formation.

General conclusion

A universal signal in plant development, auxin has emerged as a key regulator of lateral organ initiation at the periphery of shoot meristems. The dynamics of PIN1-mediated polar auxin transport in the epidermis generates localized auxin maxima and minima. Auxin maxima are responsible for organ initiation and associated cell fate changes. In addition, there is accumulating evidence for an important role for auxin minima in stem cell fate maintenance and in organ patterning. Whereas we have started to understand auxin regulation of new meristem formation, very little is known about how auxin contributes to meristem homeostasis. A main challenge for the future will be to understand auxin transport and biosynthesis in the inner corpus region in order to integrate auxin signaling with meristem homeostasis. It will also be required to further dissect auxin signaling crosstalk with other phytohormones and signaling pathways. The use of systems biology approaches, including quantitative live imaging and computational modeling, will probably be crucial to reach a new level in our understanding of auxin regulation of shoot meristems.

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